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EDITORIAL

THE SUCCESSFUL APPLICATION OF MEDICAL SCIENTIFIC KNOWLEDGE TO MEDICAL PRACTICE

WE are particularly fortunate in having obtained for this issue the major part of the presidential address delivered by Dr. F. A. Cleland to the Academy of Medicine.

Early in his address Dr. Cleland has set down the outstanding problem of the practitioner in a quotation which he gives from Dr. John Brown as follows:—"The great problem in our times is to know how to make the infinity of particulars, the prodigious treasures of mere science, available for practice."

The importance of unity of effort among all members of the profession in bringing about a "better understanding" and the unifying of this "infinity of particulars" and its application in the solution of medical problems have been strongly emphasized.

Dr. Cleland has dealt with the position of the general practitioner. He points out the possibilities which are his for the efficient and successful practice of his profession in a time when the demand for the specialist has been over-stepped. Some valuable advice is offered as to how he may by his own efforts maintain a place of high respect and prominence in the profession.

Finally, Dr. Cleland has described what he considers the most important qualities of person essential to the "true physician" and which combine the elements of art and science in practice.

We hope that no reader of this issue will fail to derive benefit from the broader point of view embodied in this address.

TANNIC ACID IN TREATMENT OF BURNS

FROM the Henry Ford Hospital in Detroit comes a new method for the treatment of burns*. The underlying principle is based upon the theory that the constitutional reaction seen in all severe cases is due to the absorption of some toxic substance or substances from the burned area. This reaction is evidenced by a rise in blood pressure, fever and redness which succeeds the primary shock symptoms of a lowered blood pressure, subnormal temperature and pallor. The fact that this toxaemia does not develop if all the burned tissue is carefully

*Ed. C. Davidson. Surg. Gyn. and Obs. Aug., 1925.

excised supports the theory. Truly remarkable results are reported from the use of tannic acid, which, by a tanning process, brings about a local coagulation of all devitalized tissue. That such a process does actually lessen the resulting toxaemia is proven by clinical results, and by its prompt appearance when the protective coagulum was removed for experimental purposes, in cases doing well at a time long after toxaemia normally manifests itself.

The technique is very simple, and consists in applying dry sterile gauze dressings which are then soaked with a 2.5 per cent. freshly prepared aqueous solution of tannic acid. In order to prevent deep caustic tissue injury, small sections of the dressing are opened for inspection after 12, 18 and 24 hours. As soon as the part is found to have assumed a light brown colour all dressings are removed. In order to facilitate removal without pain to the patient, or further traumatism, the gauze is soaked with a fresh tannic acid solution for a short time previously. The wound is thereafter left exposed to the air, but is carefully protected from mechanical injury, chilling and bacterial invasion by a suitable cradle draped with sterile linen.

The tannic acid compresses followed by exposure lessen toxaemia. Analgesic properties render it a very suitable initial dressing. This treatment results in a minimum of trauma and increases the general comfort of the patient, the wounds being absolutely painless. The local astringent action prevents the loss of fluid. There is no favourable nidus for secondary bacterial infection, and scar tissue formation is said to be less marked than with other methods of treatment. Finally the protective layer of coagulated protein forms a scaffold for the growth of young epithelial cells over the denuded surface.

The method has been under trial in the Hospital for Sick Children, Toronto, for several months, with results seemingly in accordance with the claims of its originator.

THE IMMEDIATE TREATMENT OF ACUTE RETENTION OF URINE

By H. W. Beecher Locke, M.B.

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Acute retention of urine is characterized by the presence of a distended bladder with inability to pass urine. The condition is one frequently encountered in both hospital and private practice and demands early and successful treatment.

The causes may be grouped under the following heads:—

(A) Mechanical.
(B) Nervous.
(C) Reflex.

Mechanical Factors in Acute Retention:

The majority of causes bringing about acute retention operate mechanically. They are varied in nature and vary in their site of operation. The obstruction may occur at the vesical outlet as by a collection of blood clot, or a tumour, or alteration in the prostatic gland. It may be in the lumen of the urethra by stricture, congestion, or spasm, or by calculus or other foreign body. If a stricture is present, some extrinsic cause may so affect it as to alter the lumen of the urethra cutting off the free flow of urine from the bladder. Periurethral abscess, effusion of urine or of blood may act in this fashion. Abscess of the prostate or of a Cowper's gland may by simple pressure on the lumen so occlude it as to cause acute retention.

Causes Due to Change in the Central Nervous System:

Acute retention may arise in association with cerebral or spinal injuries. Occasionally a hysterical form occurs in impressionable individuals. A temporary paralysis occasionally develops in the course of malaria, diphtheria or typhoid.

Reflex:

Operative procedures in pelvis, groin, rectum or along the genito-urinary tract are not infrequently followed by inability to void urine. This also may occur as a complication of strangulated hernia or suppurative appendicitis.

Symptoms:

In the absence of paralysis of the bladder musculature with loss of sensation, the symptoms of acute retention of urine are well defined. There is inability to pass urine. Pain in the region of the bladder is early and severe. There is constant but ineffectual effort to empty the

bladder. At most a few drops of urine may come away. If unrelieved, the patient's expression is one of anxiety; he becomes restless, and endeavours by his attitude to relieve the increased abdominal pressure. A definite pyriform tumour can be palpated in the midline of the abdomen between the pubis and the umbilicus. It can also be felt with a finger in the rectum. Palpation of this increases the patient's discomfort. In cases of paralysis there may be constant dribbling. If unrelieved, death in coma ensues in five to seven days time.

Diagnosis:

The determination of the cause and site of the obstruction is necessary in order that the most appropriate means of treatment may be adopted. The age of the patient furnishes a certain amount of aid in this. In young manhood gonorrhoeal urethritis and its sequalae account for the majority of cases of acute retention of urine by prostatic abscess, congestion or stricture. In men of fifty years or over, prostate and urethral stricture are the two most frequent causes.

In certain cases, *e.g.*, in traumatic rupture of the urethra or in post-operative cases, the history alone suggests the cause of the retention. In all cases the history should be enquired into, with particular regard to gonorrhoeal infection, past or present. Has there been frequency, nocturia, increasing hesitancy or difficulty in micturition.

Physical examination should include the whole urinary tract starting above with examination of the flanks and palpation of the renal angles. The suprapubic region is palpated, the perineum, scrotum, penis glans and prepuce are examined. Any discharge from the meatus is noted. It will be found that examination with a finger in the rectum is the most informative manipulation that can be employed. One should follow a definite routine in making a rectal examination. The patient assumes the knee chest position. The anus is inspected for fissure or other cause of perineal irritation. With the gloved lubricated index finger the tonus of the anal sphincter is tested, as definite loss of tone should direct one to a closer examination of the central nervous system. Abscess of a Cowper's gland should be excluded by approximating the index finger in the rectum to the thumb outside on each side of the midline. Immediately inside the sphincter the membranous urethra is explored against the under surface of the symphysis pubis. The finger is then carried up and a general examination of the prostate made, noting the size and consistence. Each lobe is palpated, and size, shape and presence of softening or induration noted. Care must be taken not to mistake a distended bladder for an enlargement of the prostate.

With the finger, distinction may be made between the enlargement of simple adenomyoma and the hard, nodular, irregularity of carcinoma of the prostate. Localized tenderness, increase in surface temperature and fluctuation may make the diagnosis of prostatic abscess apparent.

Treatment:

Very frequently as a preliminary to a more particular method of treatment, it is advisable on being called to a case of acute retention of urine to administer an enema of quite hot water, place the patient in a

hot bath, urging him to strive to empty the bladder and bowel while in the water. The efficacy of this routine is often such that by the time instruments are prepared the necessity for their use has ceased to exist. Such treatment usually suffices in those cases due to spasm or inflammation associated with acute posterior urethritis. If successful, a quarter grain of morphia hypodermically should then be administered and the patient put to bed. Should this not be successful in cases that are associated with acute posterior urethritis, the urethra should be thoroughly washed out with 1/5000 Oxy Cyanide of Mercury solution and a gum elastic bougie passed. Acute retention is the only condition in which one is justified in passing an instrument along an acutely inflamed urethra, and it should only then be done after cleansing the urethra as described.

The Prostate as a Cause of Acute Retention:

Inflammation.—In the course of acute gonorrhoeal urethritis, prostatic abscess is the most frequent cause of acute retention of urine. If such abscess has been determined to be present by rectal examination, the occurrence of chills and elevation of temperature, it is evacuated by perineal operation as soon as possible, and it is found that micturition is re-established as soon as this has taken place. It has been recommended to try and secure drainage of the abscess per urethra by passing a short beaked sound into the prostatic urethra, then suddenly and forcibly turning the beak to the side of the abscess. This manoeuvre should not be employed.

If operation cannot be carried out immediately, a catheter should be passed to relieve the distension and frequent hot rectal douches employed until operation is possible.

..*New Growths.*—In the presence of prostatic enlargement, whether benign or malignant, the gland may quickly undergo change sufficient to bring on acute retention. Such change may be initiated by a chill, an indiscretion in food or drink, or it may occur without obvious cause.

In the immediate treatment of such a patient, it must be appreciated that the retention which is now complete has been present in part with distension or without distension (residual urine), so that though immediate catheterization is indicated the emptying of the bladder should be a slow process. One should go to such a case equipped with a soft rubber catheter, a coudé or bicoudé gum elastic catheter, and a silver catheter with a wide prostatic curve. These should be tried in order, and if without success resort may be had to aspiration of the bladder a thumb's breadth above the pubis, using a spinal puncture needle. Twelve to fifteen ounces of urine should be withdrawn, and the puncture wound protected with cotton held in place by collodion. This will relieve the patient of his discomfort. Should the bladder be distended, a catheter provided with a cork should be tied in and a few ounces of urine withdrawn every hour until in the course of four to five days it is completely emptied. During this time, four to five pints of water should be taken by mouth daily. The bladder once emptied should not be allowed to refill before operation.

Stricture Causing Acute Retention:

If stricture is the underlying cause there is usually a fairly definite preceding history suggesting it. Under these circumstances the sudden congestion of an already partially obstructed canal explains the complete occlusion. This is brought about most frequently by acute urethritis, alcoholic excess, exposure to cold, etc. Acute retention may follow the use of too many sounds inexpertly used in the dilatation of a large calibre stricture.

The initial treatment described above should be employed; hot bath, rectal suppositories with endeavour to micturate in the bath. Failing this, beginning with number two or three French, pass gum elastic bougies. If one passes easily, increase the size until the bougie is firmly gripped by the stricture. If this enables the patient to pass urine, do not further manipulate for two or three days. If such a bougie will not pass, try a filiform bougie equipped with screw, so that a gum elastic catheter or bougie can be attached and passed through the stricture with the guidance of the filiform. If there is difficulty in passing a filiform, four to six more should be passed down to the face of the stricture and there manipulated serially until one finds the aperture. If the filiforms are not provided with thread screws for followers, tie the bougie in once it has passed the stricture and put the patient to bed. The urine will trickle along the side of the bougie, and with the relaxation of the stricture induced by the presence of the bougie it will be found that each succeeding day larger bougies can be passed until natural micturition is possible. If the stricture is impassable, a suprapubic puncture should be done, a hot bath should be given, an opiate and hot drink administered and the patient put to bed. Further effort should be made later to pass filiforms; if still unsuccessful, repeat under anaesthesia, either sacral or general.

If all these manoeuvres have been unsuccessful and the retention has existed for a number of hours, do not delay operation.

If acute urethritis complicates the presence of stricture, purge, hot baths and sedatives should be tried before any instrument is passed; and, as pointed out, this last should only be done after washing out the urethra with antiseptic solution.

Foreign Body:

Foreign body introduced into the urethra either from above or below is rarely a cause of acute retention. A calculus may lodge in the posterior urethra, when it should be gently pushed back into the bladder with a full sized sound and dealt with later. At the distal end of the urethra it may usually be extracted with forceps after meatotomy. To remove from the bulb, external urethrotomy may be necessary.

Reflex:

Catheterization should not be done until after the failure of various other expedients in those cases of acute retention that occur reflexly following operation. Change of position from recumbent to upright, or to all fours, should be tried. If this brings no success, a hot bath and belladonna and opium suppository should be administered. In the case

of infants or young children, hot fomentation over the suprapubic region usually suffices.

Acute retention may follow injury to the urethra in fracture of the bony pelvis or direct violence to the perineum. History of such injury with the issue of blood from the meatus calls for the passage of a soft rubber catheter. If this cannot be done after fair trial, suprapubic cystotomy should not be delayed.

Blood Clot Retention:

Acute retention caused by blood clot connotes a haemorrhage in the bladder sufficiently great to allow of coagulation taking place. It may be from a surface vessel of the prostate, from a vesical neoplasm, or from trauma or tumour of either kidney. Whether the blood withdrawn is bright red or black, tarry and mixed with urine, furnishes an index as to whether the bleeding is progressing or has ceased. To treat the retention, a large calibre metal catheter with a large eye and a powerful piston syringe and solution with which to keep the eye open are necessary. By means of this, evacuate the clots and then wash the bladder until the return is clear; tie in a rubber catheter and force fluids by mouth. If the haemorrhage continues, immediate operative interference is indicated.

Conclusions:

(a) A thorough knowledge of the anatomy of the lower genito-urinary tract, combined with the utmost gentleness in all manipulations, is a requisite if one is to give early relief from this distressing condition.

(b) Chronically distended bladders should be decompressed slowly.

(c) The exploration of the acutely inflamed urethra with any instrument should be avoided.

(d) In acute retention the choice of one instrument over another is decided by one thing. Always use the instrument with the use of which you are most familiar.

(e) The four chief means of treatment that we have in the order in which they should be employed are:

(1) The application of heat, by means of bath or enema.

(2) The use of the catheter; included under this head are filiform bougies for use in stricture.

(3) Suprapubic puncture.

(4) Suprapubic cystotomy, or external urethrotomy.

PRESIDENTIAL ADDRESS, THE ACADEMY OF MEDICINE, TORONTO, OCTOBER 6th, 1925

FREDERICK A. CLELAND, B.A., M.B., F.A.C.S.

Medical education never before gave such opportunity to the undergraduate as it does to-day. The equipment at hand, the amazing facilities for training in every branch of medicine, and the large fields for both clinical and original research, make many of us who remember simpler times rather envious. But the problem for the graduate when the prop of his school is removed still remains as it was when Dr. John Brown wrote over sixty years ago: "The great problem in our times is to know how to make the infinity of particulars, the prodigious treasures of mere science, available for practice." The old relation of apprentice to master which Dr. Brown goes on to picture in its better form, might very well be taken as a model for the relationship between the medical practitioner and this society. It is pre-eminently a function of our corporate body to foster the art even more than the science of medicine and to stimulate interest in the most efficient methods of practice. If the Academy developed in this way, the Fellows might avail themselves of a veritable post-graduate clinical education, apprenticed not to one master, but to many; and it is along this line we must develop.

Our aim should be to co-ordinate all the activities that are seething in laboratories, clinics, hospitals and private practices. The Academy cannot itself offer facilities for scientfic research, but it can be a clearing house and repository of the treasures of scientific adventure and discovery. We must continue to make this society so valuable a factor in the promotion and dissemination of medical knowledge throughout this city and district that no practitioner with worthy professional ideals can afford to be an outsider. We do not need a campaign to increase our membership more than we need one to interest the present members in the corporate body. Some men have the rare gift of growing wise by their own efforts, but most of us need the touch and encouragement of other minds and the spur of contact with master observers, which bring inspiration to those who in the routine of overwrought lives may easily lose the scholarly ideals of their early graduate days.

Apart from the co-ordination of medical activities, our fellowship should be concerned in fostering a better understanding between the different branches of the practising profession, in blending and assimilating our relationships, so that instead of frequent antagonisms and jealousies, we shall have just and fair co-operation. A better understanding is necessary to the settlement of certain differences which have arisen between general practice and specialism. A new condition exists, and as yet it has not developed ethical regulations. We must adjust our minds to the new conditions which the demands of the public

*Dr. Cleland's address, as given here, has been considerably abridged through deletion of several paragraphs relating to the immediate business of the Academy and the recent death of the president, Dr. McKeown.

have brought about, so that among ourselves we will do neither injustice nor disservice; we ought not to forget the antiquity of specialism, nor lose our sense of proportion in observing its growth. If we seem to be suffering from an overgrowth (I am speaking locally) we must remember that Toronto has become a very large city and a great medical centre. The public has recognized the superior service often rendered by men who concentrate in one department of their profession. It is not medicine alone that has been attacked by an epidemic of specialism, for in every profession, in law, in engineering, even in divinity, we find the same division of effort occurring, a special man for a special service. We may call the public fickle, but ultimately its demands tend to raise the standard in every occupation. The public has made specialism in medicine possible by recognizing and being willing to pay for better service. I maintain that it is just as willing to pay the general practitioner if he measures up to his opportunities. As one who for a good many years was in general practice under the most arduous conditions, I speak with much sympathy and some knowledge. The general practitioner has been the subject of sentimental comment for many years. It is said he is becoming extinct and his dirge has been sung with some bitterness and considerable misunderstanding. Ladies and gentlemen, we know perfectly well the general practitioner will never become extinct. He is a crying need, and if he fails he will have himself largely to blame. There are as great opportunities in this city for general practice as ever there were, and as much compensation in financial return and in affectionate and grateful esteem. But just as long as the general practitioner fails in continuing to educate himself along broad, sound lines, specialism of the rampant, commercial kind will feed upon him. Very little extra study work and training makes him able to handle successfully much practice now done by specialists. As a matter of fact, the number of cases in which a general practitioner requires the advice of a consultant are relatively few. He must, however, be constantly striving to attain a high standard of professional learning; but when he has not the skill nor expert information requisite to the case in question, the successful, sagacious man soon calls some one who has. He should not fail in his duty under any circumstances to provide his patient with the best possible advice and treatment. There is no loss of dignity in asking advice, and we should not forget that the general practitioner knows much that the consultant does not. The inherent weakness of most doctors, practitioners or specialists, is their unwillingness to admit they do not know. I believe this is more generally true in the medical than in any other profession. Somehow egotism seems to grow like a weed among us.

It is impossible for any man to know all branches of medical science thoroughly or to have time to practise them. No one questions the great advances in medicine which have resulted from contributions by men working in special fields. There is no division of interests between wise practitioners and those specialists who are thoroughly grounded in the fundamental knowledge of medicine, and whose art has been ripened by the wisdom of experience. This relationship is governed by old, well-established rules. It is based on integrity and courtesy, and has primarily

the best interest of the public in mind. Difficulties introduced into this relation are almost certain to be the fault of the consultant, and the physician has ample means at hand to correct them; but there is on the other hand a widespread phase of specialism peculiar to our immediate time upon which the general practitioner very justly looks with disapproval, and which calls for our grave consideration and discountenance. Sir William Osler says, "No more dangerous members of our profession exist than those born into it, so to speak, as specialists. Without any broad foundation in physiology, or pathology, and ignorant of the great processes of disease, no amount of technical skill can hide from the keen eyes of colleagues defects which too often require the arts of the charlatan to screen from the public." This is a situation which medical educationists throughout the entire world should observe with apprehension. I have been appalled in recent years in questioning students in the graduating class at our University to find that more than half inform me that they are going to be specialists of some kind or other. The complexity and economic pressure of our lives to-day tend, unfortunately, to develop a commercialism which largely explains this desire to specialize. This Academy, as a voice of the practising profession, should be ready when the time comes to give advice from its mature judgment as to how this free-lance development is to be regulated, and how a code of ethics may gradually be evolved for the guidance of our universities in instructing specialist graduates in their obligations to the profession as a whole.

The harmony and good feeling which exists in this Academy is a subject for congratulation. We have reason for gratification when in looking back over eighteen years we observe the success that has resulted from a concerted effort toward this end. Old disagreements of factions have been too well known to be supposed never to have existed. They were always unworthy, and perhaps more illusory than real, but often lurked in our minds and threw suspicion upon just criticism, and distrust upon the fairest differences of opinion. Their day has passed. We will not in this Academy admit even the existence of such a thing as a university group, an outside group, a hospital group or any other group. We are a large and cosmopolitan and broadminded fellowship. Almost all difficulties which do arise from time to time are not new. They are so old that they have been made use of by satirists in nearly all ages. Surely it would tend to tranquillize most medico troubles if we remembered how age-worn they are. Fundamentally, the cure of all real or imaginary ills in our guild lies in character; but, unfortunately, character cannot be guaranteed in a medical diploma. It is, therefore, imperative for this corporate body to emphasize and reiterate the ideals and sacred principles which have stood the test of time among the disciples of Æsculapius.

In the development of our professional character can we ever find nobler watchwords than those of Hippocrates, enunciated over two thousand years ago as essential qualities in a good physician: learning, sagacity, probity and humanity. They may be used equally by all members of our cult, by the general practitioner, by the specialist, by the investigator, by the teacher, by the custodians of public health, and

by every other to whom our door is open. Learning is not mere information, but wide and systematic knowledge acquired by reading and instruction and the result of long and assiduous study. Sagacity is rather more in the modern spirit. It is a kind of prophetic common-sense; the readiness to forsee the result of any action, especially upon human motives or conduct; and is that essential quality that leads to success in actual practice. Without probity our fraternity disintegrates; our honour is trodden in the earth. What sorrier sight than the medical doctor who possesses learning, sagacity and even humanity, but lacks probity. In no occupation or profession is strict conformity to the written and unwritten laws of justice and honour so absolutely necessary as in ours. In no calling are there such pitfalls for the man who is not guided by strictest honesty—honesty with his fellow, honesty with his patient, and, most important, honesty with his own conscience. Humanity is the groundwork, the reason of our whole structure. Without humanity no man is a true physician. Not only must he be versed in human nature, but he must be humane. He must be merciful, sympathetic, gentle, charitable; otherwise what is his justification for undertaking the treatment of human beings in an endeavour to prevent or relieve suffering. Frequently what a patient needs, perhaps, more than expert demonstration of his ills, is humane understanding and the support of a warm heart.

To acquire these four essential qualities in a good physician is a life's work. Petty necessities must never be allowed to crowd into the place of any one of them. Our profession is consecrated to a high purpose. Let us be ever watchful to repudiate the inferior practices which creep into our ranks. If evil exists let us drag it to the light. Let this Academy of Medicine write these watchwords in golden letters on her walls—Learning, Sagacity, Probity and Humanity.

PERINEPHRIC ABSCESS

T. Allen Sweet, '26.

Definition:

A perinephric abscess is a localised collection of pus about the kidney consequent upon a suppurative inflammation involving the fibrous, cellular and fatty tissues surrounding that organ.

Anatomical Considerations:

The kidneys are situated in the posterior part of the abdomen, one on either side of the vertebral column, behind the peritoneum, and surrounded by a mass of fat and loose areolar tissue. Their upper extremities are on a level with the upper border of the 12th thoracic vertebra; their lower extremities on a level with the 3rd lumbar. The right kidney is some 1 centimetre lower than the left. The kidney, with its adipose capsule, is enclosed in a sheath of fibrous tissue continuous with the subperitoneal fascia and termed the "fascia renalis." At the lateral border of the kidney the renal fascia splits into an anterior and a posterior layer. The anterior layer is carried medialward in front

of the organ and its vessels, being continuous over the aorta with the corresponding layer of the opposite side. The posterior layer extends medialward behind the kidney, and blends with the fascia on the quadratus lumborum and psoas major muscles. Through this the fascia is attached to the vertebral column. Above the suprarenal gland the two layers of the fascia renalis fuse and unite with the fascia of the diaphragm; below they remain separate and are gradually lost in the subperitoneal fascia of the iliac fossa. Numerous trabeculae connect the fibrous tunic with the renal fascia, and are strongest near the lower end of the organ. Behind the fascia renalis is a considerable amount of fat constituting the so-called "perinephric body."

The anterior surface of the right kidney is in relation to:—(1) The right suprarenal gland, (2) liver, (3) descending portion of the duodenum, (4) right colic flexure, (5) the small intestine.

The anterior surface of the left kidney to:—(1) The left suprarenal gland, (2) the spleen, (3) pancreas, (4) stomach, (5) left colic flexure.

The posterior surface of both kidneys with:—(1) The left diaphragm, (2) medial and lateral lumbo-costal arches, (3) psoas major, (4) quadratus lumborum, (5) tendon of the transversus abdominis. The right kidney lies on the 12th rib, the left on the 11th and 12th ribs of the corresponding side. Frequently the muscle fibres of the diaphragm are deficient over a triangular area, and then the perinephric areolar tissue is in direct contact with the diaphragmatic pleura.

Bacteriology:

In order of frequency the causative organisms are B. coli, streptococci, staphylococci and bacillus tuberculosis. The gonococcus, pneumococcus, B. typhosus, B. influenzae less commonly figure.

Pathology:

Infection may reach the perinephric tissue by the blood stream, lymphatics, or direct extension. The lymphatic vessels of the kidney form three plexuses—(1) in the renal substance, (2) beneath the fibrous capsule, (3) in the peri-renal fat. The second and third freely communicate, and the latter drains directly into the upper lateral aortic glands. The collection of pus is usually unilocular, being situated outside the fibrous capsule, either inside or outside the perinephric fascia. In the former case the pus will track along the ureter into the pelvis, while in the latter it will appear on the surface of the body over the iliac crest, or pass into the iliac fossa. It has been customary since the time of Rayer in 1879 to classify perinephric abscesses as (I) Primary and (II) Secondary. The case is considered primary only when the urinalysis is negative. Minute metastases and tears in the cortical layer are often doubtless precursors of seemingly primary abscesses. Of cases observed in the Mayo clinic in the past ten years not a single instance of primary infection of the peri-renal tissue resulted in abscess formation, independent of primary renal or extra-renal infection. Recent work tends to show that infection travels via the blood stream; and since the blood supply of the perinephric tissue is independent of that of the kidney, infection may be deposited there without otherwise affecting that organ. As more careful analyses are made, the relative frequency of

primary cases lessens. In 43 cases of Israel 35 were secondary. Most perinephric abscesses are due to suppuration within the kidney (Choyce 25%—Mayo 1914-1924—45%), resulting from calculus, pyelitis, pyelonephrosis, metastatic abscesses, and tuberculosis. They may also result from infection of the appendix, of the pelvic organs in women, and even of perivesical and peri-prostatic connective tissue spaces. Abscesses of the liver, gall bladder, psoas sheath and ulcerations of the duodenum may be the origin. Occasionally it arises from the extension of a pleural empyema or from Potts' disease of the spine. Trauma is a common cause with or without injury to the kidney. In a metastatic way it may arise in the course of measles, pneumonia, typhoid, influenza, or tonsillitis. Jordon, Albrecht and others have reported numerous cases from peripheral infections such as furunicles, felons, severe eczema.

Four varieties are described:

I. Above the kidney—frequently connected with intra-thoracic suppuration. The kidney is pushed down.

II. Below the kidney—may rupture into and pass along the psoas sheath pointing in Scarpa's triangle, and pass into the pelvis and rupture through the sciatic notch.

III. In front of the kidney—rare—may rupture into the peritoneal cavity, bowel, bladder or vagina.

IV. Behind the kidney—commonest of all. May pass through the lumbar muscles at the triangle of Petit.

Incidence:

The condition may occur at any age. Men more commonly affected than women (Mayo equal), and the right side more commonly than the left. Only 9 cases of bilateral are on record.

Signs and Symptoms:

In secondary cases the symptoms belonging to the primary disease precede those of the perinephritis. The beginning of the perinephric abscess is evidenced by local pain, tenderness in the loin, and more or less grave systemimc febrile disturbances. In a primary case the disease may be ushered in by chills, followed by a sharp rise of temperature and a rapid development of the local condition. The course of the blood picture and temperature are characteristic of suppuration elsewhere. The pain is often the first local symptom, and is, as a rule, more severe than in intra-renal suppuration. The pain is in the loin and costo-muscular angle, and commonly referred to the thigh and scrotum, or labia. Occasionally it is referred to the knee. The spine is, as a rule, fixed and deviates toward the affected side (this feature is easily brought out if the patient is asked to pick up an object from the floor). Psoas spasm is common, and extension therefore of the thigh is painful. The progress of the symptoms is usually rapid, so that within 10-14 days a well-marked fluctuation is felt in the loin. When the abscess is toward the upper pole, a subphrenic abscess is simulated in that limitation of diaphragmatic movement on that side is marked. Persistent vomiting, jaundice, and constipation may be present; (the higher up the original focus the more severe the alimentary symptoms). The important

diagnostic sign is local swelling. It is commonly preceded by a marked and painful resistance revealed by bimanual examination. There are three cardinal features to the swelling—(1) It is usually behind the kidney, and even when very large does not approach the abdominal wall; (2) the enlargement is diffuse, and not well defined as in enlargement of the kidney proper; and (3) it is uninfluenced by respiration. Aspiration may always be resorted to for diagnostic purposes. The pain is increased by movement, respirations, coughing and sneezing. There may be transient paralysis of the lower limb. In tuberculous cases acute symptoms are absent—pain and tenderness slight. In subacute cases pus may not form for three to six weeks.

Prognosis:

In Köster's cases the mortality was 34%; in Mayo's (1914-24) 17%. The greater the delay in surgical intervention the worse is the prognosis. In secondary cases it varies with the original cause. Experience has shown that those associated with renal tuberculosis and renal cortical abscesses are less favourable than other types.

Differential Diagnosis:

(1) Pleurisy—pain usually radiates to the shoulder, and other physical signs are present. (2) Subphrenic abscess (an encapsulated subphrenic peritonitis)—very difficult to differentiate—by aspiration, presence of an extra-abdominal cause, and no definite reaction of the lumbar muscles. (3) Intra-renal suppuration—more definitely defined, moves on respiration. (4) Haemorrhage into the kidney bed—rise in temperature does not correspond to the pain and physical signs. (5) Typhoid—no leucocytosis, Widal, blood culture. (6) Hip joint disease—not only extension but movements in all other directions equally increase the pain. Local tenderness over the joint. Pneumonia, appendicitis, cholecystitis, pyelitis, and osteomyelitis of the vertebra may simulate a perinephric abscess superficially, but with careful examination should be easily differentiated.

Treatment:

Since the time of Hippocrates, in 460 B.C., the treatment has been that of free and adequate drainage as soon as the presence of pus is established. The best incision is the oblique one through the skin, muscle layers and retro-renal fascia. A needle is often first used to locate the pus. A very careful examination of the abscess cavity is essential to determine the presence of pockets, which may require a secondary incision in the inguinal or gluteal regions. As a rule, an effort should be made to expose the kidney to view or touch. If the condition of the patient is bad it is best to defer relief of the primary kidney condition. Primary nephrectomy in the febrile state of the patient should never be done. If the drainage is not efficient, early chronic cicatrizing perinephritis may evidence itself at a considerable period after the acute perinephritic infection and the kidney may atrophy.

THE AULD DOCTOR*

DAVID RORIE, M.D.

O' a' the jobs that sweat the sark
Gie me a kintra doctor's wark,
Ye ca' awa' frae dawn till dark,
    Whate'er the weather be, O!

Some tinkler wife is in the strae,
Your boots are owre the taps wi' clay
Through wadin' bog an' sklimmin' brae,
    The besom for to see, O!

Ye ken auld Jock o' Windybarns?
The bull had near ca'ed oot his harns,
His een were blinkin' fu' o' starns,
An' doon they ran for me, O!

There's ae guid wife, we're weel acquaint,
Nae trouble's kent but what she's taen't,
Yet aye she finds some new complaint,
    O' which I hae the key, O!

She's had some unco queer mishaps,
Wi' nervish wind and clean collapse,
An' naethin' does her guid but draps—
    Guid draps o' barley-bree, O!

I wouldna care a docken blade,
Gin her accoont she ever paid,
But while she gi'es me a' her trade,
    There's ne'er a word o' fee, O!

Then De'il hae a' thae girnin' wives,
There's ne'er a bairn they hae that thrives,
It's aye the kink-hoast or the hives
    That's gaun to gar them dee, O!

Tak' ony job ye like ava!
Tak' trade, the poopit or the law,
But gin ye're wise ye'll haud awa',
    Frae medical degree, O!

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GLOSSARY—Sark, shirt; in the strae, in child-bed; sklimmin', climbing, besom, a woman of loose character; harns, brains; een, eyes; starns, stars; unco, very; girnin', whining, complaining; kink-hoast, whooping-cough; dee, die.

SOME IMPORTANT POINTS IN THE ANATOMY OF THE SEVENTH CRANIAL NERVE

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Summary

The Facial or Seventh Cranial Nerve may be considered as consisting of a motor and a sensory division. The former, besides supplying the superficial facial muscles, the "muscles of expression", also provides secretory fibres to the submaxillary and sublingual salivary glands, and probably also to the lacrimal gland.

The sensory division brings back to the brain impulses of taste derived from the anterior two-thirds of the surface of the tongue.

Motor Division

The nerve-cells of this division of the nerve form a nucleus which lies deep in the reticular formation of the pons, some distance from the mesial sagittal plane of the brain-stem. Their axonal processes run in a dorsomesial direction until they come to lie immediately under the floor of the fourth ventricle. In this part of their course they lie to the mesial side of the nucleus of the abducens or sixth cranial nerve. From here they psas dorsal to the sixth nerve nucleus, and by doing so, form a prominence on the ventricular floor known as the "facial eminence". The fibres then turn ventrally, and sweep round the lateral side of the sixth nerve nucleus to reach their point of exit from the pons.

The motor nucleus of the facial nerve has an important connection with the cerebral cortex. In the lowest part of the precentral gyrus are nerve-cells, the axonal process of which run downwards through the internal capsule and mid-brain to the pons. In the reticular formation of this part of the brain-stem they cross the mid-line, and end by forming synaptic junctions with the nerve-cells of the facial motor nucleus.

The effects of injury to the facial nerve before it leaves the brain-stem vary in accordance with whether the destructive agent affects the nucleus or the fibres which connect the nucleus with the cerebral cortex. These two types of injury are known respectively as "nuclear" and "supranuclear" lesions.

A "nuclear" lesion will cause paralysis of the muscles of the face on the affected side, because of the destruction of the motor nerve cells, the axons of which supply these muscles. In view of the intimate anatomical relations between the motor nucleus of the facial nerve and the fibres of the sixth nerve, as described above, a nuclear lesion must inevitably destroy these fibres, and so cause a paralysis of the sixth nerve. The signs, therefore, of a typical "nuclear" lesion of the seventh nerve are paralysis of the muscles of the face on one side, and inability to abduct the homolateral eye.

A "supranuclear" facial lesion produces a completely different picture. On general neurological principles it might be expected that such an injury would result in a unilateral spastic paralysis of the facial muscles, as the "upper motor" or cortico-nuclear neuron is injured. The effect typically observed, however, is that, although the lower half

of the face, involving the orbiculanis oris group of muscles, is paralysed, the upper half of the face, which corresponds to the circumocular group of muscles, shows little or no paralysis. In addition to this fact it can often be noticed that, although the lower facial muscles may be paralysed to ordinary voluntary movement, e.g., the pursing of the lips required in whistling; in violent emotional states, such as laughter and weeping, these seemingly paralysed muscles may move as well as the unaffected ones of the opposite side.

There are two explanations for these facts. The first takes as its basis the belief that each facial nucleus receives fibres from the face area of both cerebral cortices, and therefore that no unilateral supranuclear lesion could cut off all connection between the hemisphere and the facial nucleus.

The second view starts with the well accepted neurological law that the nerve-cells controlling groups of muscles which normally act together, must be very intimately connected by tracts of nerve-fibres within the nerve tissue. Applying this law to the facial muscles, it is seen that the "upper" or circumorbital group acts with the oculomotor group of muscles, and therefore one would expect intimate connection in the brain-stem between the nuclei of the third, fourth and sixth nerves on the one hand and the facial nucleus on the other. Similarly the "lower" or circumoral group of muscles acts normally in conjunction with the tongue muscles, and so one would expect a connection between the twelfth nerve-nucleus in the medulla and the facial nucleus. It is therefore believed that the facila nucleus cells can be activated both by impulses coming down the brain-stem from the oculo-motor group of nuclei, and also by impulses coming up the brain-stem from that of the hypoglossal nerve. Neither of these nerve-connections need be interfered with by an injury which interrupts the cortico-nuclear neuron of the facial.

Now the voluntary action of pursing up the lips to whistle requires for its performance the integrity of the cortico-nuclear neuron, and hence such an action cannot be performed in the presence of a supranuclear facial lesion.

The facial movements ,however, which occur in the emotional states are part of a widely diffused motor discharge, which affects not only the facial muscles, but also those of the thorax and abdomen, and it is reasonable to assume that the facial nucleus can be activated under such conditions through those connections with the motor nuclei of the midbrain and of the medulla, which have just been described.

Finally, it must be admitted that neither of the above explanations accounts satisfactorily for all the facts of "supranuclear" facial nerve lesions unless one assumes, on purely theoretical grounds, that the connections of the facial nucleus with the nervous mechanism of the eye musculature are very much more perfect than those with that of the tongue.

The fibres of the seventh nerve which carry secretory impulses to the submaxillary and sublingual glands are believed to arise in a nucleus of nerve-cells placed in the upper part of the pons, which, on account of its function, is known as the upper salivatory nucleus. These axons join the axons from the motor nucleus proper, and leave the brain-tissue in company with them.

The motor axons of the facial leave the brain-stem by emerging between the lowermost fibres of the pons. Immediately lateral to them is the point of entry of the sensory fibres of the same nerve, while further lateral is the place of attachment of the eighth nerve to the brain-stem.

The nerve then runs horizontally outwards to reach the internal acoustic meatus, which it enters, accompanied by the eighth nerve and the small internal auditory branch of the basilar artery. In this part of its course it may be injured by its being involved in organizing bloodclot on the surface of the pons, and (ii) by the pressure of tumours in this region. Tumours arising from the adjacent eighth nerve are somewhat common conditions, and the facial may be found at operation to be tightly stretched over the surface of the tumour. It is remarkable, in view of their contiguity to the facial nerve, that facial paralysis is not found to be a more common complication of the presence of these growths than is actually the case.

At the bottom of the internal acoustic canal the facial nerve becomes separated from its accompanying eighth nerve by entering a foramen which conducts it to the bony "facial canal" or "aqueduct of Fallopius", in which it runs until it emerges from the skull through the stylomastoid foramen. The nerve runs outwards in this canal until it reaches a position between the inner wall of the middle-ear cavity and the inner wall of the mastoid antrum. Here it changes its direction to run vertically downwards to the stylomastoid foramen. The rectangular bend of the canal is called the "geniculum" and in its wall are found numerous foramina. One of these foramina concerns us at present, because it transmits a branch of the motor division of the nerve which enters the middle ear cavity and supplies the stapedius muscle. Paralysis of the stapedius muscle causes hyperacusis, in which condition the person is hypersensitive to the hearing of high-pitched sounds. The presence of hyperacusis will therefore mean that the motor division of the facial nerve has been injured proximal to the geniculum.

The region of the geniculum and the subsequent vertical course of the nerve through the petrous bone is of great practical importance because of its close proximity to the middle ear cavity and the mastoid antrum. It is this part of the nerve which is apt to be damaged when these cavities are opened surgically for the removal of pus in cases of middle-ear disease. If the nerve is injured in the vertical part of the facial canal the result will be a paralysis of the facial muscles without hyperacusis, as the branch to the stapedius muscle will be intact.

The motor division of the nerve immediately after its exit from the skull through the stylomastoid foramen gives off its posterior auricular and digastric branches. The former supplies the retrahens auriculam and the occipital part of the occipito-frontalis; the latter innervates the posterior belly of the digastric and the stylohyoid muscles.

The main trunk of the nerve breaks up in the midst of the parotid gland into its two terminal divisions—the temporo-facial and the cervico-facial, which further subdivide and run horizontally forwards to supply the facial muscles. Incisions into the parotid gland are made horizontally in order to avoid damage to the branches of the facial nerve as they are running forward through the gland.

The secretory fibres of the facial to the submaxillary and sublingual glands leave the main nerve trunk just above the stylomastoid fora-

men, while the nerve is lying in the vertical part of the facial canal. They go to form a portion of the fibres of the chorda tympani nerve, and will be more fully considered with reference to the sensory division.

Sensory Division

The end -organs of taste in the anterior two-thirds of the tongue are connected with nerve-fibres which run backwards in the lingual nerve. In the pterygo maxillary fossa these fibres leave the lingual to from a separate nerve, the chorda tympani. It must be remembered at this point that the chorda tympani nerve also contains efferent fibres which are secretory to the submaxillary and sublingual glands. These run into the lingual nerve from the chorda tympani, and are distributed to these glands through the lingual and via the submaxillary and sublingual ganglia. An injury to the chorda tympani nerve will result, therefore, not only in loss of taste in the anterior two-thirds of the tongue, but also in a certain amount of dryness of the mouth due to a diminished secretion of the two salivary glands supplied by it.

The chorda tympani nerve runs upwards in the pterygo maxillary fossa, and enters the temporal bone through a foramen at the inner end of the Glaserian fissure. A short bony canal conducts it to the anterior end of the outer wall of the middle ear cavity. It runs from before backwards, lying under the mucous membrane of the outer wall of the cavity near its roof. It crosses the handle of the malleus in its backward course, and is consequently placed at the level of the upper part, the pars flaccida, of the tympanic membrane. The nerve leaves the middle-ear by passing through a foramen placed in the posterior part of its outer wall and called the iter chordae posterius. From here it is conducted by a canal to the facial canal, which it joins a short distance above the end of the latter at the stylomastoid foramen.

The taste fibres are now carried upwards in the facial nerve to the geniculum or bend of the facial canal, which marks the junction between its vertical and horizontal portions. Here the fibres reach their nerve-cells which are collected into a ganglion, which, on account of its position, is named the geniculate ganglion.

The central processes of these nerve cells carry the taste impulses onward to the brain, and together form the sensory root of the facial nerve. They enter the internal acoustic canal in company with the motor fibres, and enter the cranial cavity through the internal acoustic meatus.

Still in close association with the motor root of the nerve, the sensory fibres run horizontally inwards to become attached to the brain stem immediately lateral to the point of exit of the motor fibres.

The connections of the taste-fibres within the brain stem are still somewhat obscure. It is believed, however, that all incoming taste-fibres which will include those entering by the glosso-pharyngeal nerve, end in a nucleus of nerve-cells in the pons, which is, therefore, called the gustatory nucleus. It seems probable that the axons of these cells travel upwards through the midbrain to end in the cortex of the uncus of the hippocampal gyrus. The anatomical proof for the presence of such a tract of fibres is however lacking, and the hippocampal uncus is presumed to be the centre for taste, as well as smell, principally because it occasionally happens that lesions of that part of the cortex cause hallucinations of taste as well as hallucinations of smell.

X-RAY THERAPY IN DERMATOLOGY*

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Dermatological therapeutics honours X-Ray with a prominent position in its list of curative agents, but I am not of the opinion that it is a cure-all.

Electro-magnetic vibrations have become a very important factor since their discovery, and we find their application in wireless telegraphy, radio telephony, telephony on light beams, the therapeutic application of the Sun's ray, the rays of the Quartz or Alpine light, X-Ray and the Gamma rays of Radium. These electro-magnetic waves vary enormously in length, and the characteristic effect on our senses or on matter depends upon the wave length, not upon the speed, which is the same for all wave lengths, *i.e.*, one hundred and eighty-six thousand miles per second. It is the difference in the wave length that produces the differentiation by our senses. The unit of measurement is the Angstrom Unit, which is one hundred millionth of a centimeter. The Hertzian waves measure from an inch to several miles; the infra-red or heat waves vary from 7500 to 300,000 Angstrom units in length; the visible spectrum wave lengths are from 7500 to 3800 units long, which includes the long or red waves and the short or violet waves. The ultra violet ray of the Quartz light is as short as 1500 units. The shortest waves belong to X-Ray and Radium, and these vary from 600 to 1/10 Angstrom units.

Time does not permit me to dwell on X-Ray physics, although this is very important. All data is based on work with the Coolidge tube and a machine powerful enough that only one-half to two-thirds of its capacity is used, and this permits of greater possibilities of accuracy in the constants, that is, less fluctuation. We know the rays are obtained from the tube by the electrons striking the target or anode, and the glass of the tube wall acts as a filter to some extent; but this is left out of the picture as a factor in this discussion. But we must know that there are scattered rays produced by the substances the ray strikes in its path, as tissues or filters. These secondary rays may be therapeutic or otherwise. In deep therapy filters are used to protect the skin from the soft, long, easily absorbed wave lengths, and a filter is chosen which does not in itself give off long wave lengths.

It has been fairly well proven that in superficial therapy either filtered or unfiltered rays give about the same therapeutic effect if similar units are adhered to; but omission of the filter shortens the length of time of exposure necessary to get the resolution of the lesion, or an erythema if that is desired. This is quite an important factor. By using short exposures or fractional treatment, say, once a week, in preference to massive dosage once a month, I believe more benefit is evidenced in the lesion, and there is far less chance of any harmful after effects.

A more comprehensive view of any type of therapy is surely open to us if we study the physiological as well as the pathological effect of

*This paper was read before the section on medicine of the O.M.A. at Windsor in June, 1922, and demonstrated with lantern slides.

our agent, and then try to analyze what we are treating as viewed from its pathological aspect. No definite proof exists that X-Ray is a stimulant to cell growth, but it is thought to be. We do know that a certain dose will have an inhibitive action on the proliferation of certain types of cells. An analysis of the lesion we have to treat gives us the following data on which to base our judgment of dosage. Acute and subacute inflammatory tissues are characterized by leucocytes and round cells; these are the least resistant of all pathologic cells and the most easily influenced by therapeutic agents. Chronic inflammatory tissues are characterized by fibroblasts or connective tissue cells, and these are more resistant than the round cells. Granulomas are characterized by giant, plasma and epithelioid cells, and are less readily influenced than either acute or chronic inflammatory cells. The cells of neoplasms are less readily influenced than any of the foregoing, and they vary in their resistance, according to the tissue type giving them origin; the glandular or epithelial type being the highest, these are the most resistant. Normal tissue cells are more resistant than pathologic, but they also vary somewhat as to the age of the cell, age of the person, origin and location on the body. Cells while undergoing division are more vulnerable than in other stages of growth.

It is unfortunate that the skin of animals will not afford us data to standardize X-Ray dosage; nevertheless many excellent methods have been worked out. As time will not permit discussion of this topic, I shall dwell only on the indirect or Arithmetical computation of dosage. This method is worked out by studying the effect of the ray on the skin when using constants of spark gap or voltage, milliameter reading and anode skin distance and time. It is found that with these constants set a definite erythema will develop, and this has been called one unit. As this unit is largely the result of the work of MacKee, we will call it the M unit for the time being. There is a slight variation in tubes, but the operator should test his tube for the erythema dose; and the usual constants are M.A. 3, S.G. 6 inches, time two minutes, and distance eight inches; and if the exposure is made on a flexor surface the erythema will develop in a few days, up to ten days. By splitting the time factor various fractions of the erythema dose may be obtained. The following rules are to be remembered:

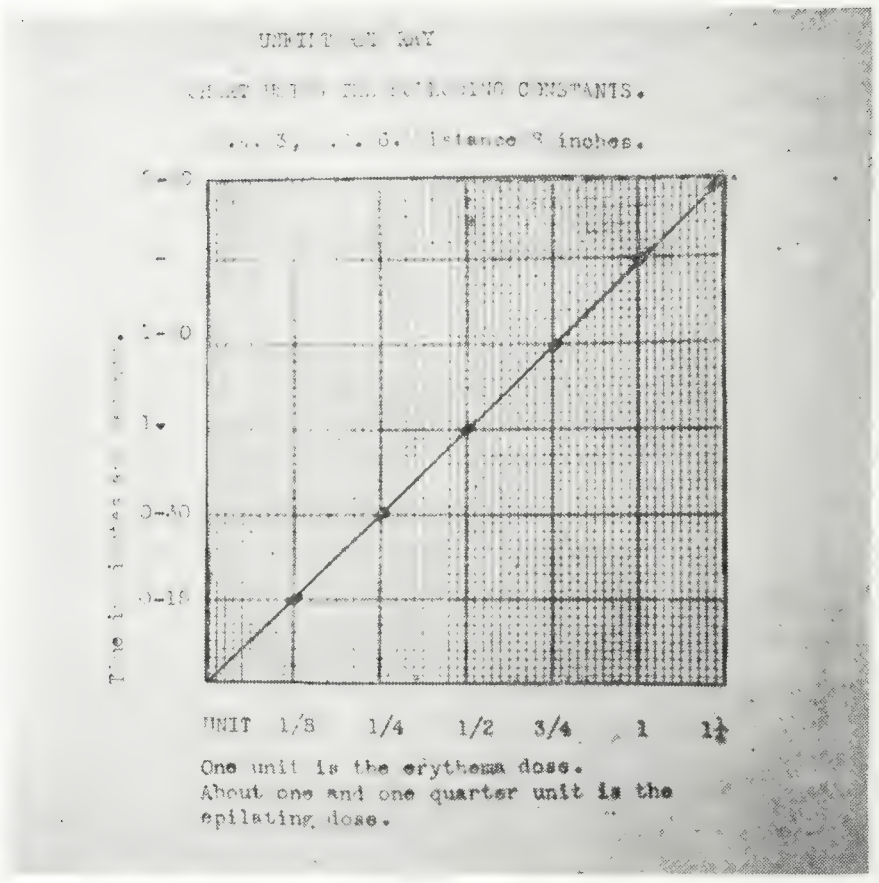
1. Doubling the spark gap doubles the dose.
2. Doubling the M.A. doubles the dose.
3. Doubling the time doubles the dose.
4. Doubling the distance gives one quarter the dose.

These rules apply only to Superficial therapy and unfiltered ray.

Working formula, $\frac{\text{M.A. } 3 \times \text{S.G. } 6 \times \text{Time } 2\text{ mins.}}{\text{Distance squared } 8 \times 8} = \frac{36}{64}$. 36/64 is known as the standard formula, and variations in any of the constants are made subject to the following rules:

A. For determination of the intensity or quantity, divide the product of any formula by the product of the standard formula.

B. For determination of the time, divide the product of the standard formula by that of the new formula.



Dosage chart made up with given constants

EXPOSURE.	SUB-FRACTIONAL.	FRACTIONAL.	SEMI-INTENSIVE.	SUB-NORMAL.	NORMAL	INTENSIVE.	MASSIVE.
Intensity.	1/16 to 1/4	1/4	1/2	1	1 1/4	1 1/2 to 2	2 to 2 1/2
Intervals.	Not over 1/4 per week.	1 week	2 weeks	4 weeks	when erythema disappears.		
Pathological Process.	Acute Inflammation.	Superficial subacute inflamation.	Simple inflammation, chronic in time. [illegible] with [illegible] chemia.	Granuloma, [illegible].	Resembling Neoplasms.	NEOPLASMS.	
Clinical Conditions.	Scalp therapy. Acute Dermatoses. Furunculosis.	Psoriasis Acne.	Thickened Psoriasis, Neuro-dermatitis, Atrophic lichen planus.	Chronic [illegible]. Seborrhea. Eczema. Keloids. Psoriasis ← Furuncles (stage)	Normal ← Sarcoma. → Spindle ⟶ Lymphoma ← Nasal [illegible] cell Epithelioma. →		Spindle celled epithelioma. →

Therapy chart giving the intensity advisable in given pathological processes and the interval between treatments and also the clinical conditions in these groups

Working charts may be prepared, and if followed, the treatment will be constant and efficient in skin lesions that respond to X-Ray therapy. This manner of dosage measurement does not apply to Quartz light therapy, in that the dosage in Quartz light therapy is increased as the skin becomes accustomed to it.

After a study of the effects of the ray on various types of cells, it is found that the response is most marked and lasting in the cells of least resistance, and that these cells do not completely recover during the treatment interval. The response is least marked and of shortest duration in the cells of greatest resistance, and these cells completely recover during the interval. The rational basis of treatment rests on the assumption that it is not the agent, but the response of the living cells to the agent, that is responsible for the therapeutic result; and the least amount of ray that will give this result is to be sought, because if the dose is too great, damage will be done to the normal cells and phagocytic cells; while if too small a dose is given, the pathological cells will recover during the interval. From a consideration of these facts three pathways are open to us in the treatment of a case. In the first we endeavour to stimulate the normal cells, as in eczema. In the second we devitalize the cells as in some chronic inflammations, granulomas or new growths. In the third we destroy, or as nearly destroy as will permit of regeneration. This is exemplified in epilation for the cure of ringworm of the scalp, not forgetting that X-Rays are not bacteriocidal to any extent. All phases of a case must be taken into consideration, and a careful study made of the cause of the lesion, whether it be constitutional or external in origin. These causes must also be treated, but it is unwise to use any of the keratolytic agents in combination with X-Ray treatment. Do not treat a skin disease with X-Ray just because it is a skin disease.

The diseases of the skin that will respond to X-Ray therapy may be classified according to location, or according to the type of pathologically active process. The latter will perhaps explain the rationale of therapy to more advantage and give us our reason for using it. We can subdivide into seven classes—(1) Simple inflammations, (2) chronic inflammation with lichenification, (3) granulomata, (4) new growths, (5) dystrophies, as keloid, (6) functional diseases, as neurodermite, and (7) epilation and the reduction of functional activity as in acne.

ON THE PATHOLOGY OF BRIGHT'S DISEASE

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The term "Bright's disease" is best used only in a clinical sense to denote renal insufficiency as evidenced by abnormal constituents in the urine and the accumulation of retained waste products in the blood. Numerous other well known signs and symptoms, such as oedema, high blood pressure, uremia, etc., frequently accompany this condition. A

variety of pathological lesions may bring about this symptom complex, and in the majority of cases the etiology is unknown.

All cases of septic inflammation, whether due to injury or to embolic or ascending infection, are excluded from Bright's disease. In such cases the etiology is clear and the condition is rather a surgical one. The term "Bright's disease" includes a variety of conditions of unknown etiology, any one of which may bring about renal insufficiency. As the etiological factors in these lesions are discovered, we may hope to arrive at a satisfactory classification.

Since our knowledge regarding the mechanism of secretion is almost as meagre as that regarding the etiology, we must content ourselves with a division based upon anatomical changes. Even such a classification is far from satisfactory, for as the lesion progresses, it often becomes impossible to determine which is cause and which is effect.

Historical study shows us that many of the earlier workers, in their zeal to demonstrate some particular fact regarding the pathology of Bright's disease, did not properly correlate their findings with other reactions taking place simultaneously in the same organ. As a result, we have lesions in different parts of the secretory unit looked upon as distinct processes. This, in addition to complicating our terminology, gives us an inaccurate idea of what is actually taking place in the kidney.

We must keep in mind that in the vast majority of cases, if one portion of the excretory unit is involved, then other parts are affected to a greater or less degree. Moreover, these processes vary, constantly influenced by conditions which depend upon a large number of outside factors and on an equally large number of ever changing individual reactions on the part of the organism.

In using the terms "acute" and "chronic" in connection with the pathology of renal disease, we should bear in mind that no sharp line of separation can be drawn between them. However, it is convenient to group kidney lesions in this way with the understanding that many intermediate forms exist. In a clinical sense the terms "acute", "subacute", and "chronic" are used to describe roughly the time limits of nephritis. In a great many cases, however, these terms do not describe the pathological state in the corresponding kidney.

In a case of severe bichloride poisoning, we can assume that we are dealing with an acute process, both clinically and pathologically. Again, in a child with a previous negative history who develops an acute attack of nephritis following scarlet fever, we may believe that such a case is pathologically acute; even here, though undoubtedly the case is clinically acute, pathologically we may find evidence of a chronic process in addition to the acute lesion. This is especially true in later life, where the kidneys, subject to an insidious chronic process, are able to "compensate", and give no clinical sign of disease. Suddenly, due to some hyperphysiological strain or a superimposed infection, we have produced a clinical case of acute nephritis, whereas pathologically we have evidence of both acute and chronic processes.

As yet there is no unanimity of opinion among pathologists regarding the manner of development of the lesion in Bright's disease. Numerous experimental studies have shown that certain toxins are able to produce

various degrees of degeneration in the epithelial lining—the secretory structure—without producing the reaction of inflammation. Just how far these lesions are to be considered inflammatory is a debatable question. In the liver the same problem arises. Are we to classify these changes in parenchymal cells as purely degenerative or to include them as inflammatory?

Any decision on this point at present is more or less an arbitrary one. However, in view of the fact that such degenerations can occur quite apart from changes which we recognize as characteristic of inflammation, and since such cases in general tend to form a clinical group which, if they recover from the acute attack, will recover completely both clinically and pathologically, it is perhaps wise to preserve a distinction between the degenerations on the one hand and inflammations on the other. Adami and Nicholls suggest that when inflammation supervenes upon degeneration, secondary infection may be the etiological factor.

Acute Bright's Disease

1. Degeneration (acute nephrosis).

Degenerations in the kidney are grouped under the term "nephroses". In this division, therefore, are grouped the various stages or types of degeneration. Such lesions are not strictly inflammatory in nature, and hence are distinguished from nephritis.

These changes may range in severity from such mixed types as albuminous or granular degeneration and cloudy swelling to fatty degeneration, and in the most severe cases necrosis of the epithelium. The character of the degeneration will depend on the type and severity of the poison, as well as on the period of time over which the injurious agent acts. In acute nephrosis the poison acts intensely for a short time and then is removed. By far the commonest causes of acute nephrosis are the toxins produced in the various acute infectious diseases, such as pneumonia, diphtheria, measles, scarlet fever, typhoid fever, etc. Fortunately these, as a rule, produce only mild degrees of degeneration, and with the disappearance of the disease and the removal of the toxin we have complete recovery, both clinically and histologically. Among the commoner poisons producing the most severe lesions are the salts of the heavy metals, especially mercury and phosphorus, and the toxins produced particularly in eclampsia, burns and the acute septicaemias. In these more severe cases we may have extensive degeneration involving the whole secretory unit, even to necrosis of the epithelium and the deposition of calcium salts in necrotic cells. This latter change occurs most commonly in severe bichloride poisoning. Thus, we see that in mild degrees of nephrosis, the epithelium, particularly of the convoluted tubules, is swollen and more coarsely granular than normal. With more severe poisons, other areas of the tubule are involved, and we may have the presence of fat. With still more pronounced lesions we have disintegration, and necrosis throughout the tubule.

The appearance of the kidney varies with the degree and extent of the degeneration. The kidney may be considerably or only slightly enlarged. The capsule strips easily. On section the kidney is pale, due

to swelling of the cells and the consequent obliteration of the capillaries. The cortex is thicker than normal, and the cortical markings are indistinct.

II. Inflammation (acute nephritis).

We have seen that in nephrosis the process is purely degenerative in character, the characteristic signs of inflammation being absent. On the other hand, in all forms of nephritis, inflammation is present. Often it is a predominant factor in the morphology of these lesions.

Many theories have been advanced as to the etiology of acute nephritis, and no doubt many factors play a part. The conviction, however, seems to be growing that the great cause underlying this condition is bacterial infection. Such factors as exposure to cold, alcoholism, debility, etc., are probably operative only in lowering resistance, and thus providing an opportunity for bacteria to gain a foothold. Whatever the true explanation as to the etiology, we do know that acute nephritis is most commonly associated with infections in the nose and throat, especially the tonsil. It may also accompany the acute infectious diseases, especially scarlet fever, and not infrequently diphtheria and typhoid fever. In each case, though, at times certain portions of the secretory unit appear to be affected more than others, the tendency in all cases of nephritis is to produce a diffuse inflammation throughout the kidney.

Let us now consider the pathological changes in the different tissues in acute nephritis, at the same time remembering that each is involved in the same process to a varying extent.

Malpighian Bodies:

Herring has shown that the cells lining Bowman's capsule, those covering the glomerulus, and those of the convoluted tubules at an early period in their development are morphologically similar; but during further growth they become specialized, probably for the performance of three different functions. These different areas also react differently in disease.

In acute inflammation, one of the earliest changes observed is a swelling in the cells covering the glomerular tuft. The glomerular tuft early commonly shows increased cellularity. There are three possible sources of origin for these cells. Kelsch and Keiner considered them to be epithelial, Klebs that they are a proliferation of the connective tissue lying between the capillaries, while Councilman and Welch consider this increase to be the result of proliferation of the capillary endothelium, and to a certain extent increase in connective tissue cells. Leucocytes may also be present in large numbers. Later the swollen epithelial cells may desquamate into the capsular space. Various types of thrombi may be found in the glomerular capillaries. Councilman and Klein have described hyaline and fibrinous thrombi in acute inflammatory conditions.

Bowman's capsule also undergoes changes. Greenfield has pointed out the importance and extent of the cellular proliferation inside the capsule. The capsule itself may also be swollen. There is sometimes a considerable accumulation of albuminous exudate between the tuft and

the capsule in the capsular space. This exudate may also contain fibrin, leucocytes, and red blood cells.

Tubules:

The glandular elements of the kidney are mesoblastic in origin. On account of the anatomical arrangement of the blood supply, there is an intimate relationship between changes in the glomerulus and changes in the corresponding tubules. The exact relation of these changes is difficult to estimate. The tubular changes are probably due to the combined effect of nutritional disturbance and the direct action of the agent bringing about the glomerular lesion. The lesions in the tubules are largely degenerative in character and resemble very closely those described under "nephrosis". The epithelium of the convoluted tubules is chiefly involved. In more severe cases the distal convoluted tubules and loops of Henle are also affected. The epithelium is swollen and coarsely granular in the milder types. In more severe cases we have fatty degeneration, desquamation, and disintegration of the cells. The lumina of the tubules may then contain detritus of a varied character. Desquamated and disintegrating epithelial cells, serum, red blood cells and leucocytes may be seen, all of which aid in the formation of the various types of casts.

Interstitial Tissue:

Normally this is very small in amount, being most abundant between the ducts of Bellini in the medulla. The amount of change here varies considerably. In the early stages, oedema is usually present, and there may also be a varying number of leucocytes seen. In all acute inflammations, accumulations of round cells, especially around the glomeruli and larger blood vessels, are to be observed. Councilman considers that these cells are derived from the lymphoid cells of the blood. General thickening of the interstitial tissue may occur even in acute nephritis, particularly in the type following scarlet fever. The blood vessels are also involved in the inflammatory process.

In the gross such kidneys appear almost normal. They are, however, moderately enlarged. The capsule is thin and stretched tightly over the surface. The capsule strips readily and the cut margins of the cortex evert. The kidney surface is usually reddish or mottled reddish and grey. The stellate veins are frequently engorged. The cortex is thicker than normal, and the colour varies considerably. It is pale and cloudy, presenting a marked contrast to the dark red medulla. The glomeruli are frequently seen as little reddish dots. Minute petechial haemorrhages can be seen scattered over the cortical surface and throughout its substance. The extent of the changes described varies greatly in different cases. The exact cause of these variations at present is not known.

If the changes are most prominent in the glomeruli, the term "acute glomerulo-nephritis" has been used; if in the interstitial tissue, "acute interstitial nephritis"; if in the tubules, the term "acute nephrosis with nephritis" may be used. However, these finer pathological differences cannot be differentiated clinically; and the important feature regarding this non-suppurative inflammation is the diffuseness and tendency to involve simultaneously the various parts of the kidney structure.

(To be continued)

SIR MICHAEL FOSTER

BERNARD WILLINSKY, L.D.S., D.D.S., '28

Sir Michael Foster was born in Huntingdon on March 8, 1836. He was the eldest son of Michael Foster, F.R.C.S., a surgeon practising in that town—a man of marked character and held in much esteem. He was brought up in an atmosphere of fervent nonconformity in religion, and of strong radical leanings in politics. His early education was carried on at Huntingdon Grammar School, and later (1849 to 1852) at University College School in London. From the first, Foster showed considerable aptitude for study, doing well in the usual routine of school work, and before he left University College he became captain of the cricket team. Cricket was the only game to which afterwards he had the least inclination. At Cambridge he arranged an annual cricket match, to which in the beginning of things, he invited all those working in his laboratory, and for some years the match was a high laboratory festival.

At the early age of 16, Foster matriculated at the University of London, entering in the same year at the University College. He distinguished himself in most of the subjects of the Arts curriculum, and in 1854, in the London University Honours Examination for the degree of Bachelor of Arts, he took first place in Classics, and was awarded the Classical Scholarship. Having taken this degree, he was face to face with the necessity of choosing a profession. Hitherto his training had been entirely on the Arts side, but in view of the opening in life afforded by his father's practice, he chose medicine. So in 1854 he began attendance on the preliminary scientific and medical lectures of University College, and at once, or almost at once, entered on clinical work at the hospital. His versatility was shown by his carrying off the Gold Medal for Anatomy and Physiology, and that for Chemistry. In 1858 he took the M.B. degree of the University of London, and in the following year the degree of M.D.

The next two years were partly spent in further medical study—for which purpose he spent some time in Paris both in 1859 and in 1860—and partly in original investigation.

About this time Foster showed indications of pulmonary disease, and adopted the usual remedy of the time—a sea voyage. The steamship Union was about to proceed to the Red Sea, to build a lighthouse on the Asarugi rock opposite Mt. Sinai. He obtained the post of Surgeon, and on the voyage utilized the time making observations on the marine fauna.

In 1861 he returned to Huntingdon, and joined his father in practice. Two years later, he married Miss Georgiana Edmunds. His married life was brief, his wife dying in 1869, leaving a son—Dr. M. G. Foster of San Remo—and a daughter. Whilst occupied with the varied cases that come to a medical practitioner in a country town, he kept in touch with Physiology. He wrote an account ,still worth reading, on the theories of the coagulation of the blood.

In 1867 Foster abandoned medical practice and went back to University College, London, where he was appointed Teacher of Practical Physiology, and also performed the duties of Curator of the Museum of Pathology. Not long after, when Dr. George Harley, who held the

Professorship of Practical Physiology and the Lectureship of Medical Jurisprudence, was unable to discharge his duties on account of illness, they were performed by Foster, and in 1869, Foster was elected to the Professorship. In the same year, he was appointed Fullerian Professor of Physiology at the Royal Institution.

Foster's genial nature and attractive personality laid the foundation of many lasting friendships, both amongst the younger and those older than himself.

In 1870, Foster was thirty-four years of age, and though from early manhood he had kept the pursuit of science steadily in view, he had been in a large degree the shuttlecock of circumstances. But at this time he obtained an appointment which gave free play both to his natural qualities and his varied experience. The University of Cambridge, with its scientific traditions, was becoming aware of the gradual rise of branches of science in which it took little or no part, and it was ripe to bear the impress of any exceptional personality. The first step came not from the University itself but from Trinity College. The College founded a Praelectorship in Physiology, and elected Michael Foster Praelector. No election could have had more fortunate results for the University.

The University gave to the Trinity Praelector a room, now part of the Philosophical Library, for use both as a lecture room and as a laboratory. Trinity College supplied the simple furniture, some tables,—three of which put together at the top of the room, served to mark off a space which served for the lecturer's platform—some bottles and reagents, a few microscopes, and so on. It is hardly an exaggeration to say that from this room there arose, by successive extensions, nearly all the laboratories now devoted in Cambridge to Biological studies.

The primary idea of Foster's teaching was that scientific instruction should be based upon firsthand knowledge, and that a student must see and do things for himself in order to gain a real and lasting hold on any scientific subject. Foster did not underrate the value of reading, but he insisted that descriptions of facts and discussion of theories could be only imperfectly comprehended apart from laboratory work.

Foster in his other chief interest had still greater and more immediate success. The majority of the small band who attended his lectures during the first three years adopted a scientific career. Of these Gaskell, Balfour, Dew-Smith, Sheridan Lea, and Langley would probably not have followed science but for Foster's influence. His influence was not exerted by way of exhortation. The desire to undertake research was imbibed from him in lecture and in familiar talk. It was the air they breathed. He was of the widest catholicity as to the kind of research to follow. Of his other pupils at this time, Liversidge became Professor of Chemistry, and Milnes Marshall, Professor of Zoology. His numerous pupils in later days were always assured of his sympathetic attention. As Professor Sherrington has said, "He took a genuine interest in younger men and their work, and he rejoiced in their scientific achievements often with an almost paternal delight."

In 1871 the honorary degree of M.A. was conferred on Foster by the University, and in the same year he was elected a Fellow of Trinity College. In 1872 he married Miss Margaret Rust, of Huntingdon, who became his constant companion both at home and in his annual holiday,

in earlier years in Switzerland, and in later years in Venice. In 1883, consequent on the Report of a Royal Commission, a Professorship of Physiology was founded in Cambridge, and the Trinity Praelector of Physiology, without any practical change of duties, became Professor in the University. In the following year the complete degree of M.A. was conferred upon him. He was a member of the Council of the Senate from 1886 to 1890. He did not take any great part in the ordinary routine of University business, but he had a keen interest in the establishment of new studies, and the Department of Agriculture, at its inception, had in him an ardent supporter. In 1900 his increasing duties outside the University led to his applying for a Deputy to perform the duties of the Professorship, and in 1903 he resigned his office.

In Cambridge it will not be forgotten that the modern developments of Biological Science in the University are primarily due to Sir Michael Foster. He transformed the method of teaching, extended the scope of the subjects, and implanted higher ideals. And it should be remembered that his influence—exerted both directly, and indirectly through his pupils—extended far outside the limits of the University. The scientific study of Physiology, Botany and Morphology in English-speaking countries is largely indebted to him for some of its best features.

Foster wrote many articles on scientific subjects for reviews, and was in constant request for addresses and lectures. Of the latter one or two may be mentioned. He delivered the address on Physiology at the meeting of the British Medical Association at Cambridge (1878); and at the meeting of the International Medical Congress in London (1881). He delivered also one of the inaugural addresses at the International Medical Congress at Rome (1894). He gave the Rede Lecture at Cambridge in 1893, and a course of lectures at the Cooper College, San Francisco, in 1900. The latter course of lectures he issued in book form in 1901 as "A History of Physiology during the 16th, 17th and 18th centuries"—a scholarly work, written in an easy, picturesque manner.

It might also be mentioned that he visited Toronto in 1897, as President of the Physiological section of the British Association for the advancement of science.

His scientific work was interfered with materially by his dabbling in politics, it seems a shame that a man of his great ability should become interested in this field of endeavour, where a truly analytical scientific mind as he had would really be out of place.

In still other ways than these, Foster's initiating spirit was of notable service to Physiology. He founded the *Journal of Physiology*, and took a large part in promoting the establishment of the Physiological Society and of the International Congress of Physiologists.

Shortly after his second marriage he built a house on a spur of the Gog-Magog hills, about four miles from Cambridge. There on a bare chalk slope backed by a spinney of low growing trees he laid out a garden, which soon became the favourite afternoon resort of his many friends. His garden was his chief relaxation. He loved masses of flowers, and had large beds of cyclamen, anemones, daffodils, irises and many others. His rock garden in early summer was a blaze of colour. He was especially successful in growing varieties of Eremurus, and their noble spikes were a great feature of the garden. But he loved still more growing

rare plants and experimenting in hybridization. Most of all he loved irises, and many a traveller in distant lands enriched his collection. On these he became one of the first authorities, and produced a number of new hybrids, with an account, on which he was busy to the last.

Foster received numerous distinctions in recognition of his scientific services. He was honorary or corresponding member of many learned societies both at home and abroad. He received honorary degrees from the Universities of Dublin, Glasgow, Montreal, Oxford and St. Andrews, and in 1899 he was created Knight Commander of the Order of the Bath.

Foster suffered little from ill health during the greater part of his life. The early symptoms of tuberculosis disappeared. He was an early riser, and spent as much time as he could spare in the open air in his garden. Though careful in diet, he only abstained entirely from those things for which he had no taste. He dined out rather frequently, and when he dined on public occasions he was commonly asked to speak. As an after-dinner speaker he was excellent, and, at his best, was in the very first rank. He spoke, as he lectured, very slowly, and without any freedom of gesture. A peculiar gleam of the eye announced the coming of a humorous allusion, and a jest was followed by an inimitable suppressed laugh which was the delight of all his friends. On other occasions he was grave and earnest and, as Dr. Pye-Smith has said, "Few public speakers knew better how to express sympathy or sorrow in dignified and graceful terms".

In 1904 he had a severe attack of dyspepsia, which almost incapacitated him for work, and for a time robbed him of his enjoyment of life. But the pain and discomfort gradually became less, and in the summer of 1906 he was again active and his cheerfulness had returned. On Monday, January 28, 1907, he made a short speech in London at a meeting of the British Science Guild, in the evening he was taken ill, and was attended by his friend Dr. Rose Bradford. In the night he died suddenly from pneumo-thorax, caused by the bursting of an oesophageal ulcer.

In summarizing Foster's work—it might be said that his actual additions to our knowledge by way of research are small and not of great importance. He was, as one of his pupils said, a discoverer of men rather than of facts and theories. A man of Foster's type fulfills many functions in the scientific world, not only by his own work, but by the powerful stimulus he offers to other workers, and through this factor many notable advances in physiology have been made, and in quoting Sir Michael Foster's own words, "For indeed it is one of the lessons of History of Science that each age steps on the shoulders of the ages which have gone before. The value of each age is not its own, but is in part, in large part, a debt to its forerunners.

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Ergot for Hypodermic Use

Some of our most valuable drugs are dependent entirely upon the pharmaceutical manufacturer for their reliability. Take ergot as an example. It is not to be expected that all natural specimens will contain the same percentage of active principle, and experience has proved that they do not. The necessity of standardizing ergot preparations has long been apparent, but chemical methods were not available because of the complexity of the active principles. Once it was thought that ergotinic acid was the active principle, but now the less of this an ergot preparation contains the higher it is rated, other things being equal. The alkaloid ergotoxin is very important, but certain amounts of the amines, histamine and tyramine, must also be present.

Since, however, ergot has long been used in medicine for its effect upon the involuntary muscles, the idea occurred to Dr. Houghton, of Detroit, in 1895, that an ergot preparation might be tested by administering it to cocks and observing its effect upon the comb, the degree of bluing or blackening produced being taken as an indication of the physiological action of the specimen. In 1898 this method was adopted by Parke, Davis & Co., as a standard method for assaying their commercial products of the drug. It is now generally recognized as the most practicable method of assay known.

It is sometimes desirable to administer ergot hypodermically, but the ordinary fluid extracts are not suitable for this purpose. To give a small dose double efficiency, a preparation is now available called Ergot Aseptic, each cubic centimeter of which is equivalent to two cubic centimeters of the official fluid extract. Further particulars are given in the Parke, Davis & Co. advertisement elsewhere in this issue.

Advt.

LACTIC ACID MILK IN INFANT FEEDING*

A. G. Smith, '26,and C. G. Shaver, '26

In introducing this paper we first of all want to draw your attention to the subject of gastric acidity, because on this point rests the rationale of lactic acid milk as a food for infants.

Considerable experimental work has been done by Marriott and others, which briefly shows the following results.

1. Gastric acidity reaches its maximum one and one-half hours after a feeding.
2. The normal acidity of gastric contents varies between a PH = 3.5 and PH = 5.0.
3. The various digestive enzymes of the stomach each have an optimum PH, at which they act best.
4. The acidity varies in health and disease.

Experimental results show that the normal range of gastric acidity is well within the zone of effective digestion.

*Presented before the Paediatric Reading Club November 20, 1925.

It has been noted that a gastric acidity of PH = 3.5-5.0 is necessary for proper digestion, because not only is the action of enzymes best at this level, but infection by harmful organisms is kept down by the acid content, and furthermore the pancreatic juices are better stimulated by an acid chyme. For these reasons, some method of regulating the gastric acidity is necessary, in order to allow for optimum gastric digestion.

Infants depend mainly on breast milk or cows milk for their nourishment. Cows milk contains buffer substances, which greatly reduce the acid of the stomach, and thereby interfere with proper digestion. There are three ways of getting around this difficulty. The first is by a simple dilution of the milk with water. The second method is by drying the milk, whereby the soluble buffer salts of calcium and phosphorus are rendered insoluble and lose their buffer action. The third means is by the addition of some acid or acid forming organism to the milk.

The first method is in general use, but not only are the buffers reduced by the dilution, but also the caloric value, and a stomach already abnormal in function is called upon to handle large volumes, which it does poorly.

Drying of the milk as advocated in the second method, depletes it of the accessory food factors, and hence renders it non feasible as an infant food.

The third method of acidifying the milk or adding to it some organsim which is acid forming, offers the most encouraging results, not only experimentally but also clinically.

It was only after an exhaustive study of the actions of the various acids on cows milk that lactic acid was found to do the work in the most efficient and least harmful manner. It is interesting to note the work of the experiments in order to ascertain why lactic acid and lactic acid forming organisms were chosen.

Faber thought that theoretically there was no reason why the acid normally found in the stomach, namely hydrochloric, should not be used. He drew attention to the fact that hydrochloric acid milk had been primarily used in the tretment of tetany with good results.

He started off using 10 per cent. HCl and found that he must add 50-60 cc. of the acid to 100 cc. of whole cows milk to get a resulting gastric acidity of PH = 5.0. The disadvantages were, the sour taste, heavy flocculations of casein, and the tendency to acidosis, as the chlorides were not readily eliminated. Most children refused this preparation because of the taste, and in preparations in which the acid was reduced sufficiently to make the mixture palatable, the resultant gastric acidity fell far below the zone of effective digestion.

Benham and Dunham attempted to find an acid mixture that (*a*) would be acceptable to the child, (*b*) would be cheap and readily obtainable, (*c*) could be easily handled at home. They finally chose acetic acid 5-6 per cent, as fulfilling the above requirements. They found that the acetates were readily absorbed from the intestine, that they did not inactivate calcium, and that they had no poisonous effect. The organic acid was completely oxidized to carbon dioxide and water, and did not have to be eliminated as had the inorganic acid.

In spite of all these good qualities, Marriott showed that milk acidified in this way often caused diarrhoea, which is a complication ever

to be guarded against, especially in those children for whom an acidified milk is prescribed.

Citric acid, in the form of lemon juice, was used by Hess. He used this acid for two reasons. Firstly, it is a normal constituent of milk, and secondly contains antiscorbutic vitamine. The strength of the mixture was $33\frac{1}{3}$ per cent. acid, which gave a resultant gastric acidity of PH = 5.3. This mixture therefore also falls below the required range of gastric acidity. It was also found that infants under two months of age could not tolerate such a mixture.

It remained for Marriott to introduce lactic acid milk. He found that lactic acid neutralized the buffers in cows milk, had no objectionable taste, had certain antiseptic qualities, and in some way increase the infant's tolerance to sugar and fat. It is a physiologic acid, is completely oxidized, and can be used at an earlier age than the other preparations.

Further work revealed the fact that milk acidified by the action of lactic acid forming organisms was more beneficial. Therefore most of the lactic acid milk in use at the present time is prepared by the addition of such organisms as B. Lacticis or Streptococcus Lacticis.

Lactic acid milk results in a gastric acidity of PH = 4.0 which is well within the zone of effective gastric digestion and compares well with the results obtained with breast milk.

Preparation

Lactic acid milk was first used in Holland in the form of buttermilk, but as this is of low caloric value, and there is no method of standardization, it gave way to a milk produced by more scientific methods.

Metchnikoff produced it by using the Bacillus Bulgaris in 4 per cent. milk. The results obtained from the milk were very good, but the organism was difficult to grow and propogate. The next organism for use that was brought to light was the Bacillus Acidophilus. This produced a very good milk, and it is still used in some hospitals in the States. The chief disadvantage is that it is difficult to propagate.

Marriott was the first man to use streptococcus lacticis; an organism that is normally present in sour milk. He found that it would grow easily, was not easily contaminated, and produced milk of such an acidity that pathogenic bacteria could not readily survive in it. Also the organism can be dispensed to almost anywhere without injuring its growth.

In The Hospital for Sick Children, Toronto, 2 per cent. milk is used instead of 4 per cent., which was the original prescription of Marriott. The method of preparation is as follows:

Remove four ounces of cream with a Chapin cream dipper from the top of one quart of sweet milk, on which the cream has risen. Boil the remainder three minutes; cool and strain. Then mix three tablespoons of culture with the cooled milk. Cover with a clean towel, and leave undisturbed in a warm room 12-15 hours. If the milk is disturbed it sets in a powdery instead of the normal junket consistency. When set, keep in a cold place. Before using beat with an egg beater until smooth enough to pour well, but not frothy. The acidity of this resultant mixture will vary from day to day.

Save three tablespoons of this lactic acid milk in a sterilized bottle as culture to make the next days supply.

If not set in fifteen hours, use more culture.

Contamination of the culture is rare, but occasionally in the summer it may be overgrown by a yeast organism, which produces enough gas to expel the corks from the bottles.

Preparation of Acidified Milk

Remove four ounces of cream with a chapin dipper from the top of a one quart bottle on which the cream has risen. Boil the remainder for three minutes; cool and strain. Measure and make up for evaporation by adding boiled water up to the original amount. To 16 ounces of this cold boiled milk add one teaspoonful of lactic acid, drop by drop, stirring continually.

Preparation of Lactic Acid Milk Powder

This is made by spraying the lactic acid milk at a low temperature, so that the organism is not killed. The result is a moist, yellowish white powder. To make lactic acid milk from the powder, mix five and one-half packed level tablespoons of the powder with ten ounces of water, beat in with an egg beater.

The caloric value of 4 per cent. lactic acid milk is 19 calories per ounce, while the 2 per cent. preparation has a caloric value of 14 calories per ounce. The taste is not unlike buttermilk, and seems to be quite suitable to the infant's appetite.

Indications for the use of Lactic Acid Milk

Lactic acid milk is indicated in those cases where a substitute for breast milk is needed; such is the case in marasmus, where the child is unable to assimilate its food. The two per cent. milk is recommended in this condition, and its attributes are due to the fact that it supplies sufficient calories as well as being easily assimilated. Sugar is added when the stools are normal. Any of the sugars may be used, but corn syrup is the one usually employed.

Those infants who are suffering from gas, colic and constipation, which are symptoms of irritation of the small bowel, do well with lactic acid milk. The results here are due to an overgrowth of the intestinal flora by the streptococcus lacticis which inhibits the process of fermentation.

In infectious diarrhoea also, the streptococcus lacticis will overgrow the intestinal tract, and kill off the offending organism.

The above indications only include diseased conditions, but there are those cases in which a child is being fed on cow's milk mixtures, and is showing a hesitating improvement. These infants if given a complete change of diet to 2 per cent. lactic acid milk, will show marked improvement in their general condition.

It is necessary to point out that acidified milk has not the wide use of lactic acid milk, because it does not contain the streptococcus lacticis which by its rapid multiplication in the intestine, brings about the desired result in cases of excessive fermentation and infectious diarrhoea; also for some unknown reason the presence of the organism exerts some favourable action.

Clinical Results

Clinical results obtained from feeding lactic acid milk and lactic acid milk powder, have been identical. The stools are putrefactive, and range from one to three per day in number.

Some results published by the St. Louis Hospital, where a lactic acid milk feeding experiment was carried out in 142 difficult feeding cases, showed the following:

1. An average of 70 calories per pound body weight per day was fed.
2. Average daily gain for the entire group was 9/10 ounces.
3. The infants rarely developed diarrhoea or vomiting, and none died as a result of gastro-intestinal upset, due to overfeeding.
4. The mortality among marasmic children, in the hospital, fell from 78 per cent. in 1919 to 26 per cent. in 1922.

Medical Society

The Medical Society Executive announce the following appointments to this years Daffydil Committee:

Chairman.....W. C. M. Scott, Vth Year
Secretary.....R. P. Vivian, VIth Year
Treasurer.....H. K. Moir, VIth Year
Editor of Epistaxis.....T. H. Belt, IVth Year
Stage Manager.....A. R. J. Heffering, Vth Year
Property Manager.....E. B. Patterson, Vth Year
Ladies Representative.....Miss Rachel K. Haight, IVth Year
Year Representatives: W. B. Brebner, VIth Year; L. A. Macklin, Vth Year; J. G. Shannon, IVth Year; J. A. Fallon, IIIrd Year; G. W. Lewin, IInd Year; J. M. Spence, Ist Year

Mr. G. F. Houser, VIth Year, has been appointed musical director for the year 1925-26.

The first open meeting of the Medical Society was held in Hart House Lecture Room, on the evening of Wednesday, November 11. Honorary Chairman, Dean Primrose, fittingly introduced the speaker of the evening Dr. N. B. Gwyn, who gave an interesting outline of the life and works of the late Sir William Osler, as a student and a teacher.

The Medical Society Executive are reviving the afternoon open meetings which were so popular in past years. The first of these was held on Wednesday, December 9, at 5 p.m., in the Anatomy lecture room. Dr. L. J. Austin, Professor of Surgery at Queen's, presented in masterly fashion a popular lecture entitled "Ambroise Paré and His Times."

Mr. W. A. McTavish, Assistant Secretary, represented the Medical Society at the Annual Engineering Society Dinner in Hart House on November 12, 1925.

Mr. W. L. Deeton, President of 2To, represented the Medical Society at the annual At-Home of the Aesculapian Society of Queen's University on Friday, November 13, 1925.

Athletic Society

Rugby

Senior Meds. came through in Rugby football with the second championship of the year for us. Considering the number of men Medicine has given to Varsity teams this year, this is a remarkable performance. Meds. are always in the running somewhere for the cup. The whole team did their work well, individually and collectively, but the backfield attracted most attention. Captain "Les" Mullen, with his kicking, and "Gordie" Large, with his broken field running, starred. Both caught with few fumbles, and these were promptly recovered. Among the line men Tom Flommerfelt and "Hip" Smith were much in evidence. "Hal." Burgess and "Hiker" Rutherford were always there when needed. Farmer engineered the games creditably. The whole team did well and deserve credit.

The following participated: Captain "Les." Mullen, "Gordie" Large, "Hal" Burgess, "Hip" Smith, Tom Flommerfelt, Farmer, Lawson, Vivian, Goodchild, Chesney, Rutherford, Irwin, Murray, Webster Angus, Bull, Caswell, James, Shaver, Aberhard, Gordon, and Fletcher.

Water Polo

In Water Polo Meds. have won their group, and are now in the play off for the Ekhardt Cup. Should they win it will be the first time Medicine has done so. The team has won five games and lost only one. Moir, Flommerfelt and Derbyshire have been continually in the spotlight.

We have two championships won, and are already in the finals of a third. Let's go and capture those of Hockey, Basketball and Boxing and Wrestling—we have the material—thus making this the most successful year yet.

Assault-at-Arms

The Annual Medical Assault-at-Arms was held in Hart House on the evening of December 3rd, 1925. Although the entry list was smaller than that of last year, some new material was uncovered, and so the purpose of the assault was accomplished. This is particularly well illustrated by the good showing made by the first and second years in tying for second place.

The Class of '27 won the cup for the second time.

The scores made by the various years were as follows:

Year	6th	5th	4th	3rd	2nd	1st
Points	4	11	0	3	6	6

Boxing

175 lbs. Chesh Dreyer, VIth Year, won from Sam Teskey, Vth Year, by a close referee's decision. Both football players, going in for the sake of their year, fought an interesting if not highly skilful bout.

158 lbs. Byrnes, 1st Year, won from Homer Bull, Vth Year, by a referee's decision. The men were evenly matched.

145 lbs. Sher, Vth Year, won from "Mo." Mitchell, IIIrd Year, by default.

135 lbs. Ziegler, IIIrd Year, won from Ken Ferrie, Vth Year, by a technical knockout. Ziegler is a new man in the ring, but was in better condition than his opponent. In the first round it looked very much as if he would be knocked out. During the second he staged a wonderful comeback, and keeping up his left, he punished severely with his right until Ferrie was completely overcome.

125 lbs. Magee, IInd Year, won from MacKechnie, IVth Year, on a referee's decision. The winner showed more aggressiveness.

118 lbs. Webber, IInd Year, won from Flemmery, IIIrd Year, by a technical knockout. The latter showed a finer style but lacked stamina.

Wrestling

Heavyweight. Gordon, Vth Year, won from Jackson, Vth Year, by two falls. Gordon had the advantage in weight.

175 lbs. Chesney, Vth Year, won by default, from Lawson, IVth Year, who had a sore hand.

158 lbs. Farnsworth, VIth Year, won by default from White, IInd Year.

145 lbs. Saunders, Vth Year, won from Doyle, IInd year. by one fall in overtime in a very interesting bout.

135 lbs. Webster, Vth Year, won from Torrance, Ist Year, by referee's decision in overtime. Torrance showed great promise as Webster is an Intercollegiate 118 lbs. champion.

125 lbs. Wilson, Ist Year, won from Webber, IInd Year, by two falls.

Softball

The 3T0 Softball Team got away to a good start in the inter-year series by defeating the crack 2T9 outfit, 1924 champions, 7 to 6, on Tuesday, December 2.

Basketball

In the Interyear Basketball competition, the speedy first year team defeated 2T9 17 to 14 in the finals.

Graduation Dance

The 2T6 Graduation Dance is being held at the King Edward Hotel on Friday, December 18. A very capable committee composed of J. G. Campbell, R. P. Vivian and S. S. Murray, are planning an interesting evening's entertainment.

The patronesses are Mrs. Alexander Primrose, Mrs. Herbert Bruce and Mrs. E. Stanley Ryerson. Music is being supplied by Herb. Smith's Orchestra.

Members of Graduating Year Ill

Two very popular members of 2T6 have been ill during the past few weeks, and we will be very glad to welcome them back to year activities soon. W. D. Robson has undergone an Appendectomy at the Western Hospital, and Irving Sparks is in the Isolation Hospital suffering from Scarlet Fever.

Soph.-Frosh Banquet

Defying all superstitions surrounding Friday the 13th, Meds. of 3T0 and 3T1 got together for a most successful Soph.-Frosh banquet on that date last month. The festivities began with a splendid feast at the Prince George, and concluded with a theatre party at the Strand.